A DISSERTATION ON

NEURO DEVELOPMENTAL ASSESSMENT AND FOLLOW UP IN ASPHYXIATED BABIES IN THE FIRST YEAR OF LIFE WITH EMPHASIS ON EVOLUTION OF TONE

M.D (BRANCH VII)

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CERTIFICATE

This is to certify that the dissertation entitled **"Neuro developmental assessment and follow up in asphyxiated babies in the first year of life with emphasis on evolution of tone"** submitted by **Dr.P.PUNITHA** to the Faculty of Paediatrics, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Paediatrics) is a bonafied research work carried out by her under our direct supervision and guidance.

Dr.P.Amutha Rajeshwari,M.D.,DCH.,

Professor and Head of the Department,
Institute of Child Health & ResearchCentre,
Madurai Medical College,
Madurai.

DECLARATION

I **Dr. P. PUNITHA**, solemnly declare that the dissertation titled "**Neuro developmental assessment and follow up in asphyxiated babies in the first year of life with emphasis on evolution of tone"** has been prepared by me.

This is submitted to the **Tamilnadu Dr.M.G.R.Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D.Degree Examination in Paediatrics.

Place: Madurai

Date: **Dr.P. PUNITHA**

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CONTENTS

INTRODUCTION

With modern methods of neonatal care, babies have begun to survive after insults which were previously thought to be fatal. As a result of this there is an increase in the number of survivors and also in the type of brain lesion for which they suffer. Hence it is becoming increasingly important to keep a close watch on the neuro development of these graduates of neonatal intensive care unit.

PERINATAL ASPHYXIA:

It refers to a condition of impaired gas exchange that leads, if persistent to fetal hypoxemia and hypercarbia.

It occurs during first and second stage of labor and is identified by fetal acidosis, as measured in umbilical arterial blood.

Birth asphyxia and Hypoxic ischemic encephalopathy (HIE) are associated with increased mortality and morbidity.

Neonatal depression is a general term used to describe an infant who has prolonged transition from intrauterine to an extrauterine environment. These infants have low 1- and 5- minute Apgar scores.

Neonatal encephalopathy is a clinical term used to describe an abnormal neurobehavioral state that consists of decreased level of consciousness with abnormalities in neuromuscular tone.

Hypoxic ischemic encephalopathy is an abnormal neurobehavioral state in which the predominant pathogenic mechanism is impaired cerebral blood flow.

Hypoxic ischemic injury refers to neuropathology attributable to hypoxia and / or ischemia as evidenced by biochemical (serum creatinine kinase brain bound [CK-BB], electro physiologic (EEG), neuroimaging, magnetic resonance imaging (MRI), computed tomography(CT), or postmortem abnormalities.

INCIDENCE:

The frequency of perinatal asphyxia is 3-5% of the live births in Indian infants. It is inversely related to gestational age and birth weight. A higher incidence is noted in infants of diabetic or toxemic mothers, infants with intrauterine growth restriction, breech presentation, and postdated infants.

ETIOLOGY:

In term infants, ninety percent of asphyxial events occur in the antepartum or intrapartum period as a result of impaired gas exchange across the placenta that leads to the inadequate provision of oxygen and removal of carbon dioxide and hydrogen ions from the fetus. The remainder of these events occurs in the postpartum period and is usually secondary to pulmonary, cardiovascular, or neurologic abnormalities.

PATHOPHYSIOLOGY:

During labour complicated by a hypoxic ischemic challenge, the following changes may occur:

1) With brief asphyxia, there is a transient increase, followed by a decrease in heart rate (HR),mild elevation in blood pressure(BP), an increase in central venous pressure (CVP), and essentially no change in cardiac output(CO). This is accompanied by a redistribution of cardiac output with an increased proportion going to the brain, heart and adrenal glands (diving reflex).

2) With prolonged asphyxia cerebral blood flow becomes dependent on systemic BP (loss of cerebral auto regulation). A decrease in CO leads to hypotension and impaired cerebral blood flow resulting in anaerobic metabolism and eventually intracellular energy failure due to an increase in the utilization of glucose in the brain and a fall in the concentration, of glycogen, phosphocreatine, and adenosine triphosphate(ATP).

3) Hypoxia induced vascular dilatation increase glucose availability, atleast transiently; and anaerobic metabolism produces lactic acid.

HYPOXIC ISCHEMIC ENCEPHALOPATHY:

The diagnosis of perinatal HIE requires an abnormal neurologic examination on the first day following birth. It is important to note that no significant neurologic abnormality diagnosed later in childhood can be ascribed to perinatal asphyxia in the absence of evidence in the immediate neonatal period of neurologic abnormality and severe multiorgan dysfunction.

HIE is associated with long term neurodevelopmental sequelae.

Overall more severe the encephalopathy greater is the risk of sequelae.There may be major sequelae like cerebral palsy, mental retardation, epilepsy, visual and auditory impairment or mild motor deficits in later life or subtle neurologic abnormalities. Even in the absence of obvious neurological deficits in newborn period there may be long term functional impairments.

Changes in neuromotor function observed during the first year of life are closely related to the maturation of central nervous system and presence or absence of brain damage. Hence it is important to detect abnormalities in the neurodevelopment as early as possible, so that intervention programmes can be started.

TONE: Muscle tone is difficult to define. It is the resistance of the muscle to stretch. It is that condition of the muscle determined by physical, chemical and nervous influences, which determine body posture, the range of movements at joints and feel of muscle. Passive tone indicates extensibility of muscle. It is observed by a maneuver evaluating the amplitude of slow movement executed by the observer, with the infant remaining passive. The result is expressed as an angle or in relation to a landmark.

REVIEW OF LITERATURE

1. **González de Dios J** et al[1] - There have been several attempts to relate either perinatal asphyxia at birth or abnormal neurological findings after asphyxia in neonatal period (hypoxic-ischemic encephalopathy), to outcome.

RESULTS: The incidence of neurologic sequelae, in 115 asphyxiated full-term infants follow-up at least 12-24 months, was 16.5%; 4 cases had severe sequelae, 4 moderate and 11 mild. The overall asphyxia-related infant mortality rate was 0.87/1000 live births. The main sequelae detected at follow-up were motor disability, and other disabilities like mental retardation, epilepsy, sensorial defects, were infrequent. The incidence of cerebral palsy was 0.87/1000 live births, and in among that 2.6% is in asphyxiated term neonates.

2. Sachin Shah et al [2] - Hypoxic ischemic encephalopathy (HIE) is the most important consequence of perinatal asphyxia. The syndrome of HIE has spectrum of clinical manifestations ranging from mild to severe, that correlates with severity of the insult. Recognition of perinatal asphyxia depends on information gained from careful history and thorough neurological examination.

Perinatal asphyxia is associated with high mortality and morbidity. However, not all survivors manifest later neurological handicaps, even following apparently severe neonatal neurological symptomatology. It would be of considerable clinical and academic value to be able to identify in the neonatal period which neurological symptomatic neonates show evidence of organic cerebral lesions and which do not, and to be able to relate such information to later neurological function.

3. Robertson CM et al [3] - Available evidence shows that adverse sequelae do not follow perinatal asphyxia unless encephalopathy is part of the neonatal clinical presentation. Where neonatal encephalopathy follows evidence of late fetal and/or early neonatal distress, the staging of the encephalopathy is useful for determination of prognosis; those with mild encephalopathy do well; those with severe encephalopathy do poorly.

4. Developmental assessment tests [4]: scopes and limitations Indian Paediatrics vol 36 july1996 - Neonatal care has made amazing advances in the last twenty years and the survival of 'high risk' infants has increased considerably.

Pediatricians have now started realizing that follow up services are an integral part of this neonatal care. Hence developmental assessment is no longer the realm of developmental neurologists or psychologists, but pediatricians also need to be familiar with the common developmental tests and at least need to understand how to interpret the results of these tests.

5. Paro- Panjan D et al[5] - Amiel-Tison Neurological Assessment at term. The aims of this study were: (1) to perform the Amiel-Tison Neurological Assessment (ATNA) in a group of infants with different risk factors for brain damage; (2) to analyze the results of the examinations in light of the risk factors and presumed etiology; (3) to compare results of examinations with results of cranial ultrasound, electroencephalography (EEG), and cerebral function monitoring (CFM); and (4) to evaluate neurological outcome at 12 to 15 months of age using the Amiel-Tison and Gosselin method, and developmental outcome using the Bayley Scales of Infant Development.

6. Paro-Panjan D et al [6] -Information provided by the neonatal neurologic assessment is important for identifying infants with neurologic abnormalities at a very early age. The aim of this study was to compare two distinct approaches to the neurologic assessment of newborns: the Amiel-Tison neurologic assessment, and Prechtl's

qualitative assessment of general movements.

The agreement of the neurologic and developmental outcome was better with the Amiel-Tison assessment (kappa = 0.39, 0.77) than with the observation of general movements (kappa = 0.38, 0.37).

7. Sudha Chaudhari et al [7] - Passive tone indicates extensibility of muscle. It is observed by a maneuver evaluating the amplitude of a slow movement executed

by the observer, with the infant remaining passive. The result is expressed as an angle or in relation to a landmark (scarf sign). The waxing and waning pattern of tone was described by Amiel-Tison based on the work of Dargassies.

8. Meherban Singh [8]: Care of the newborn 7th edition. Evaluation of muscle tone is useful for early diagnosis of cerebral palsy. During fetal life acquisition of muscle tone and motor functions evolve from lower extremities and spreads upwards in the direction of head. Healthy term babies are hypertonic by adult standards. The process of muscle tone spreads cephalo- caudally after birth. Thus the upper limbs begin to relax and acquire skills before the lower limbs. The head control appears first, followed by ability to sit, stand and finally walk by 12-18 months.

Amiel – Tison‘s method for assessment of muscle tone is useful for early diagnosis of cerebral diplegia. Reduction of these angles occurs due to hypertonia and is suggestive of cerebral palsy. Indian babies are physiologically more hypotonic possibly due to higher incidence of intrauterine growth retardation and unsatisfactory postnatal nutrition.

There are some babies who develop transitory tone abnormalities during 3-6 months of age but normalize by the age of 9 months to one year. These infants may manifest with subtle neurological abnormalities and learning difficulties later in life.

9. Fetal and neonatal brain injury Third edition Long-term follow-up of term infants with perinatal asphyxia Charlene M.T. Robertson .[9]

Follow-up of children thought to have perinatal asphyxia has added to our understanding of the proportionately small role intrapartum asphyxia plays in childhood cerebral palsy, and has clarified the low risk of this developmental motor disability for asymptomatic newborns. The value of the presence of and staging for hypoxic–ischemic encephalopathy (HIE) is widely accepted.

10. Manual of Neonatal care, 6th edition John P.Cloherty [10.] Severity of encephalopathy can be ascertained using the Sarnat clinical stages of HIE. Stage I 98% to 100% has a normal neurological outcome and < 1% mortality. Stage II 20% - 37% die or have abnormal neurodevelopmental outcomes. In one study, half of the 42 surviving infants who had Sarnat stage II encephalopathy had normal development at one year of age; approximately 10 % had a normal neurologic exam and mild developmental delay and one-third were diagnosed with cerebral palsy. Prognosis is considered to be good if an infant does not progress to and/or remains in stage III and if total duration of stage II is less than five days.

Some neurologically normal survivors of perinatal asphyxia have problems in school. In one study all stage I HIE and 65% to 82% of stage II HIE children performed at expected level at 8 years. In another study, children 8–13 years old who had neonatal encephalopathy plus Apgar score <4 had increased risk of problems with mathematics, problems with reading, epilepsy, minor motor problems, attention deficit hyper activity disorder compared to unaffected children.

11. Nelson text book of Paediatrics. 18th edition [11]: The outcome of HIE correlates to the timing and severity of the insult and ranges from complete recovery to death. The prognosis varies depending whether the metabolic and cardiopulmonary complications are treated, the infant's gestational age and the severity of encephalopathy.

Severe encephalopathy characterized by flaccid coma, apnea, absent oculocephalic reflexes, and refractory seizures, is associated with a poor prognosis. A low Apgar score at 20 minutes, absence of spontaneous respirations at 20 minutes of age, and persistence of neurological signs at two weeks of age also predicts death or severe cognitive and motor deficits.

Infants with stage II and stage III encephalopathy are at highest risk for adverse outcome. Microcephaly and poor head growth during the first year of also correlate with the injury to the basal ganglia and white matter and adverse outcome at twelve months. All survivors of moderate to severe encephalopathy require comprehensive high risk medical and developmental follow up. Early identification of neurodevelopmental problems allows prompt referral for developmental, rehabilitative, neurological care, and early intervention services so that the best possible outcome can be achieved.

12. The development of the infant and young child 10th edition Ronald S.Illingworth [12].

Estimation of muscle tone:

Muscle tone is assessed in the newborn or older infant as follows:

1. Observation of posture
2. Feeling of muscle
3. Assessing the resistance to passive movement
4. Assessing the range of movement
5. Shaking the limb
6. Indirect assessment – by tendon jerks, plantar response and moro reflex.

Spastic form:

On holding the infant in vertical suspension: there may be abnormal extension of hip and knees and the legs may cross.

In ventral suspension: There is usually delayed motor development and so there will be excessive head lag. The arms and legs commonly hang down lifelessly without the flexion of the elbows and knees and slight hip extension seen in normal child.

Some infants show good or even advanced head control in ventral suspension and in prone position, due to excessive extensor tone, and so give the wrong impression of having advanced motor development; but on pulling the child to the sitting position the gross head lag is obvious. It is incorrect to term the head lag hypotonia.

In supine position the symmetry or asymmetry
of the kick. Note whether the hands are equally open or closed.
Assess muscle tone by feeling the muscles, assessing the resistance to passive movement, assessing the range of movement, and shaking the limbs when holding the arm below the elbow and leg below the knee. Assess the range of movements in the hips and in dorsiflexion of ankle.

On pulling him to the sitting position in order to assess the head lag. Sway him gently from side to side in order to determine the degree of head control. When he is being pulled up into the sitting position, have a hand in the popliteal space in order to detect spasm of hamstrings. When a child is spastic one feels a resistance to pulling up to the sitting position and the knees may flex, one can see this and feel the spasm of the hamstrings. When leaned forwards he repeatedly falls back, because of excessive extensor tone, and give the wrong impression of advanced weight bearing. The true diagnosis is revealed by other signs of excessive tone, exaggerated reflexes, ankle clonus, and reduced abduction of the hip and ankle dorsiflexion, with head lag when he is pulled up.

Persistence of primitive reflexes beyond three months helps in the diagnosis of cerebral palsy.

AIM AND OBJECTIVES

AIM:

- To assess the neuro development in asphyxiated babies.

OBJECTIVES:

1. To detect the abnormalities in the neuro development of asphyxiated babies as early as possible.
2. To evaluate the changing measurements of tone in the form of various angles in first year of life.

MATERIALS AND METHODS

a) STUDY DESIGN:

Observational study

b) STUDY PLACE:

Institute of child health and research centre,

Govt. Rajaji Hospital, Madurai-20.

c) STUDY PERIOD:

December 2006 to June 2008.

d) STUDY POPULATION:

Babies delivered in Government Rajaji Hospital,

Madurai-20

e) SAMPLE SIZE:

227 babies

f) INCLUSION CRITERIA

- GRH delivery
- Term babies
- Birth weight : 2.5kg – 3.9 kg
- Uneventful ante natal history
- All modes of delivery
- HIE I & II

g) EXCLUSION CRITERIA:

- Pre term babies
- Birth weight : < 2.5kg & > 4kg
- HIE III
- Presence of congenital anomalies
- Presence of birth injuries
- Depression from maternal anesthesia/ analgesia
- Babies with sepsis and meningitis

METHODOLOGY

After admission in the newborn ward the babies to be studied were registered. The parents were informed about the study and their consent was taken. Detailed antenatal and birth history was taken. The infants were examined. Their weight, length, head circumference was recorded. Clinical examination was done to rule out any obvious congenital anomaly or birth injury. Cry, suck, activity and primitive reflexes were noted. By clinical neurological examination babies were grouped by Sarnat and Sarnat staging of HIE.

After discharge the babies were followed up at well baby clinic every month in ICH & RC upto the age of one year. However, formal neuro developmental assessment was done at third, sixth, ninth and twelfth months. At every visit, a detailed anthropometry was recorded. Weight, length, head circumference were plotted on a growth chart to ensure that there was no growth faltering. Nutritional advice and immunization were given.

Sarnat and Sarnat's Clinical stages of H I E

	Stage I	**Stage II**	**Stage III**
Level of consciousness	Hyper alert	Lethargic or obtunded	Stuporous
Neuromuscular control			
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive	Decreased or absent
Segmental myoclonus	Present	Present	Absent
Complex reflexes			
Suck	Weak	Weak or absent	Absent
Moro	Strong; low threshold	Weak; incomplete; high threshold	Absent
Oculovestibular	Normal	Overactive	Weak or absent
Tonic neck	Slight	Strong	Absent
Pupils	Mydriasis	Miosis	Variable; often unequal; poor light reflex
Heart rate	Tachycardia	Bradycardia	Variable
Autonomic function	Generalized sympathetic	Generalized parasympathetic	Both systems depressed
Bronchial and salivary secretions	Sparse	Profuse	Variable
Gastro intestinal motility	Normal or decreased	Increased; diarrhea	Variable
Seizures	None	Common; focal or multifocal	Uncommon (excluding decerebration)
EEG findings	Normal (awake)	Early: low-voltage continuous delta and theta Later :periodic pattern (awake) Seizures : focal spike and wave	Early : periodic pattern with isopotential phases Later : Totally isopotential
Duration	<24 hrs	2-14 days	Hours to weeks

TOOLS USED

A) Neurodevelopmental assessment

1. A red ball for visual fixation and pursuit.
2. A pooja bell to test hearing at three months.
3. A piece of paper – crackling of paper at six months.
4. A red rattle to test voluntary reach and transfer of objects.
5. A coloured paper clip to test for pincer grasp.
6. A paper and pen to see for scribbling at one year.

B) Evaluation of passive tone

Goniometer: This is a non traumatic portable instrument used for measuring angles. It consists of two arms attached with a screw.

On one arm a protractor is fixed which is calibrated upto hundred and eighty degrees.

Neurodevelopmental assessment:

1. Examination of head.
2. Neurosensory evaluation – visual fixation and pursuit were tested by a red ball.
3. Hearing was tested by a pooja bell at three months and

crackling of paper at six months.

4. Primitive reflexes – stepping and placing disappears by six weeks, palmar grasp and Moro disappears by three months and asymmetric tonic neck reflex at three to six months.

5. Protective reflexes like lateral propping and parachute reflex appears at nine months.

6. Motor milestones – Appearance of social smile, head support, sitting, crawling, pincer grasp and standing were recorded.

7. Evaluation of tone was done at resting posture, passive tone and active tone.

Spontaneous posture was observed by inspecting the child while he/ she lies undisturbed. Passive tone was evaluated by applying certain maneuvers to the infant while he remains at rest. These maneuvers must be performed slowly, gently, and just to the point of discomfort. The resistance of an extremity to this manipulation was measured by recording the angle formed at this joint by this movement, using a goniometer.

The following angles were measured.

1. Angle at hip: Adductor angle.
2. Angle at knee: Popliteal angle.

3. Angle at ankle: Dorsiflexion angle. The angle was measured using a slow and a quick movement. A difference of less than ten degrees between the slow and quick angle was considered as normal. The angles were expressed both as mean (standard deviation) and as ranges. These ranges were compared with those described by Amiel – Tison angles.

Active tone was studied with the infant moving spontaneously in response to a given stimulus like pull to sit and pull to stand.

OBSERVATION, ANALYSIS AND RESULTS

A total number of 227 babies were enrolled for the study and were followed up for one year.

Hospital admission data for seven months December 2006 to June 2007, total number of live births were 7243, newborn admission 1537, asphyxiated babies 565. Out of 565 babies after excluding babies with prematurity, birth weight <2.5 kg, HIE III, birth injuries and congenital anomalies, 227 babies were registered and followed up in well baby clinic upto the age of one year and the neuro developmental status was evaluated periodically at the interval of three months.

STATISTICAL ANALYSIS:

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2002).

Using this software, frequencies, percentages, means, standard deviations, chi square, ‘p’ and coefficient of correlation values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate’s test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship. If the coefficient of correlation (r) is more than 0.5 then the two variables are taken to be correlated.

BASELINE DATA:

Two hundred and twenty seven full term infants were prospectively followed up for a period of one year. Twenty cases lost follow up. So only two hundred and seven infants came for all the four assessments. In that there were one hundred and thirty three infants in HIE stage I and seventy four cases in stage II.

Table - 1

HIE	**I**	**II**	**Total**
Enrolled	149	78	227
Completed follow up	133	74	207
Drop out	16	4	20
Drop out %	10.7	5	8.8

Table - 2

MODE OF DELIVERY

Mode of Delivery	Cases	
	No	%
Labour Naturale	104	50.2
Forceps	62	30.0
Vacuum	3	1.4
Breech	3	1.4
LSCS	35	16.9
Total	208	100

Based on mode of delivery there were 104 cases (50.2%) delivered by labour naturale, 62 cases(30%) by forceps delivery, 3 cases (1.4%) by vacuum delivery, 3 cases (1.4%) by breech delivery and 35 cases (16.9%) by LSCS delivery.

Table - 3

Anthropometric measurements

	< 3rd percentile	3rd – 50th percentile	50th – 95th percentile
Weight	12	177	18
Length	9	170	28
Head circumference	26	144	37

Twelve babies out of 207 had weight less than third percentile at twelve months of age, nine babies had length less than third percentile at twelve months of age and twenty six babies had microcephaly.

NEURODEVELOPMENTAL ASSESSMENT

1. Examination of head: Microcephaly was present in 26 infants out of 207 infants. Among that HIE stage I babies were nine and HIE stage II babies were seventeen. Among those seventeen HIE stage II babies with microcephaly three babies had gross developmental delay and features of cerebral palsy.

2. Visual fixation and pursuit was present in 167 infants by three months, 38 infants by four to five months and in two infants not appeared till one year. Three babies had concomitant squint.

3. Hearing: Among the 207 babies, 199 babies were able to turn towards sound at three months and eight babies by five months.

4. Primitive reflexes: Disappearance of stepping, placing, palmar grasp, moro and asymmetric tonic neck reflex.

Table - 4

Disappearance of primitive reflex in	Cases	
	No	%
3 months	121	59.0
4 months	40	19.5
5 months	22	10.7
6 months	16	7.8
7 months & above	6	3.0
Total	205	100
Range	3-9	
Mean	3.78	
S.D.	1.16	

Among the 207 babies primitive reflexes disappeared by 3 months in 121 (59%), 4months in 40 (19.5%), 5months in 22 (10.7), months in 16 (7.8%), 7months and above in 6 (3%) and in two cases not disappeared by one year.

5. Protective reflex:

Table - 5

Appearance	Cases (No.)	Cases (%)
9 months	108	52.9
10 months	51	25
11 months	25	12.3
12 months	20	9.8
Total	204	100

RANGE	9-12 months
MEAN	9.76 months
S.D	1.1

Appearance of lateral propping reflex and parachute reflex by nine months in 108 babies(52.9%), ten months in 51 (25%), eleven months in 25(12.3%),twelve months in 20 (9.8%) and in three cases protective reflexes not appeared by twelve months.

6. Milestones:

Table - 6

	Age of testing (months)	Appeared	Delayed	Not appeared
Social smile	2	176 (85%)	31(15%)	-
Head control	4	177(85.5%)	28(13.5%)	2(1%)
Sitting	8	173(83.6%)	31(20%)	3(1.4%)
Crawling	10	170(82%)	34(16.4%)	3(1.4%)
Pincer grasp	10	172(83.1%)	30(14.5%)	5(2.4%)
Standing	12	169(81.6%)	31(14.9%)	7(3.4%)

Among the milestones, social smile was tested at 2 months, it was appeared in 176(85%) of babies and delayed in 31 (15%) babies. Head control was tested at 4 months, it was appeared in 177(85.5%) of babies, delayed in 28 (13.5%) and not appeared in 2 (1%) of babies. Sitting without support was tested at 8 months, appeared in 173(83.6%), delayed in 31(20%) and not appeared in 3(1.4%). Crawling was tested at 10 months, appeared in 170(82%), delayed in 34(16.4%) and not appeared in 3(1.4%). Pincer grasp was tested at 10 months, appeared in 172(83.1%), delayed in 30(14.5%) and not appeared in 5 (2.4%). Standing was tested at 12 months, appeared in 169(81.6%), delayed in 31 (14.9%) and not appeared in 7 (3.4%).

7. **Passive tone:**

The adductor, Popliteal and dorsiflexion angles were measured with goniometer.

Adductor angle: It is measured at third, sixth, ninth and twelfth month. The measurements were shown in the following table.

Table -7

Measurement at	Adductor angle		
	Range	Mean	S.D
3 months	38 – 102	68.6	11.9
6 months	43 – 124	93.2	14.5
9 months	56 – 143	119.2	16.1
12 months	63 – 153	135.0	12.4

The mean adductor angle at three months was 68.6 degree with a S.D of 11.9, at six months 93.2 and 14.5, at nine months 119.2 and 16.1 and at twelve months 135.0 and 12.4.

Popliteal angle:

It is measured at third, sixth, ninth and twelfth month. The measurements were shown in the following table.

Table - 8

Measurement at	**Popliteal angle**		
	Range	**Mean**	**S.D.**
3 months	62 – 113	90.6	7.3
6 months	74 – 134	109.4	10.8
9 months	89 – 190	132.5	14.6
12 months	62 – 169	148.4	14.6

The mean Popliteal angle at three months was 90.6 degree with a S.D of 7.3, at six months 109.4 and 10.8, at nine months 132.5 and 14.6 and at twelve months 148.4 and 14.6.

Dorsiflexion angle:

It is measured at third, sixth, ninth and twelfth month. The measurements were shown in the following table.

Table - 9

Measurement at	**Dorsiflexion angle**		
	Range	**Mean**	**SD**
3 months	48-68	61.2	3.3
6 months	53-72	64.8	3.1
9 months	53-73	67.2	3.4
12 months	55-74	69.1	3.3

The mean dorsiflexion angle at three months was 61.2 degree with a S.D of 3.3, at six months 64.8 and 3.1, at nine months 67.2 and 3.4 and at twelve months 69.1 and 3.3.

Scarf sign:

It is noted at third, sixth, ninth and twelfth month.

Table -10

Measurements at	Medial to midline		At midline		Crosses midline	
	No	%	No	%	No	%
3 mon	205	99.0	2	1.0	-	-
6 mon	16	7.7	189	91.3	2	0.9
9 mon	1	0.48	32	15.4	174	84
12 mon	-	-	8	3.8	199	96.2

The scarf sign was medial to midline at three months in 205 babies out of 207(99%), at midline in 2 babies out of 207 (1%).

At six months medial to midline in 16 babies, (7.7%), at midline in 189 babies (91.3%), crosses midline in 2 babies (0.9%).At nine months medial to midline in one baby (0.48%), at midline in 32 babies(15.4%), crosses midline in 174 babies (84%). At twelve months at midline in 8 babies (3.8%), crosses midline in 199 babies (96.2%).

Measurement of angle at infancy (stage I cases no.133) Table-11

	3 months		6 months		9 months		12 months	
Angle	**study group**	**Amiel Tison**	**study group**	**Amiel Tison**	**study group**	**Amiel Tison**	**study group**	**Amiel Tison**
Adductor								
Range	48-102	40-80	73-124	70-110	102-143	100-140	113-153	130-150
Mean	72.7		98.2		124.9		138.8	
SD	9.2		10.3		10.8		6.6	
Popliteal								
Range	64-113	80-100	92-134	90-120	112-158	110-160	125-169	130-170
Mean	92.7		112.9		137.1		152.1	
SD	6.0		7.8		10.1		12	
Dorsi flexion								
Range	53-68	60-70	59-70	60-70	62-73	60-70	62-74	60-70
Mean	61.9		65.6		68.2		69.9	
SD	2.6		2.6		2.6		2.6	
Scarf sign								
Medial %	98.5	Medial	0.8	Elbow at	-	Elbow	-	Elbow
At ml %	1.5	To	97.8	midline	0.7	crosses	-	crosses
Crosses%	-	midline	1.5		99.3	midline	100	midline

For HIE stage I cases the angles were measured using goniometer and scarf sign were seen. The values of these angles at all four ages, expressed as mean and standard deviation are shown in the table. The same angles described as ranges by Amiel – Tison are shown along side.

Table 12

Measurement of angle in Infancy (stage II cases in Study group n = 74)

Angle	3 months		6 months		9 months		12 months	
	Study group	**Amiel Tison**	**Study group**	**Amiel Tison**	**Study group**	**Amiel Tison**	**Study group**	**Amiel Tison**
Adductor **Range** **Mean** **SD**	38-82 61.2 12.8	40-80	43-108 84.2 16.5	70-110	56-137 108.9 18.9	100- 140	63-148 128.2 16.8	130-150
Popliteal **Range** **Mean** **SD**	62-104 86.8 7.9	80-100	74-127 103.2 13.0	90-120	89-190 124.3 17.6	110-160	94-168 141.9 16.4	130-170
Dorsi flexion **Range** **Mean** **SD**	48-68 59.9 4.0	60-70	53-72 63.4 3.4	60-70	53-72 65.4 3.8	60-70	55-74 67.7 4.1	60-70
Scarf sign **Medial (%)** **At Midline (%)** **Crosses midline (%)**	100 - -	Elbow medial to midline	20.3 79.7 -	Elbow at midline	1.3 43.2 55.5	Elbow Crosses midline	- 10.8 91.2	Elbow Crosses midline

For HIE stage II cases the adductor, popliteal and dorsi flexion angles were measured using goniometer and scarf sign were seen. The values of these angles at all four ages, expressed as mean and standard deviation are shown in the table. The same angles described as ranges by Amiel – Tison are shown along side.

Table - 13

Measurement of angles in Infancy (Stage I & II for study cases)

Angle	3 months		6 months		9 months		12 months	
	Study group	**Amiel Tison**	**Study group**	**Amiel Tison**	**Study group**	**Amiel Tison**	**Study group**	**Amiel Tison**
Adductor **Range** **Mean** **SD**	38-82 61.2 12.8	40-80	43-108 84.2 16.5	70-110	56-137 108.9 18.9	100- 140	63-148 128.2 16.8	130-150
Popliteal **Range** **Mean** **SD**	62-113 90.6 7.3	80-100	74-134 109.4 10.8	90-130	89-190 132.5 14.6	110-160	62-169 148.4 14.6	150-170
Dorsi flexion **Range** **Mean** **SD**	48-68 61.2 3.3	60-70	53-72 64.8 3.1	60-70	53-73 67.2 3.4	60-70	55-74 69.1 3.3	60-70
Scarf sign **Medial(%)** **AtMid line(%)** **Crosses midline(%)**	99 1 -	Medial to midline	7.7 91.3 0.9	At midline	0.48 15.4 84	Crosses midline	- 3.8 96.2	Crosses midline

Table -14

Correlation HIE staging with passive tone

Stage	Adductor angle		Popliteal angle		Dorsiflexion angle		Scarf sign % of cases		
	Mean	SD	Mean	SD	Mean	SD	Medial	At midline	Crosses midline
I	138.8	6.6	152.1	12.0	69.9	2.6	-	-	100
II	128.2	16.8	141.9	16.4	67.7	4.1	-	13.5	86.5
‘ p ’	0.0001 Significant		0.0001 Significant		0.0002 Significant		0.0001 Significant		

There was a significant correlation (p value 0.00001) established between HIE staging and passive tone.

8) **Active Tone:**

Head lag, pull to sit and pull to stand were noted from three months onwards.

Table -15

	Range (months)	**Mean (months)**	**SD**
No head lag	3-7	3.46	0.86
Pull to sit	6-10	6.36	0.82
Pull to stand	9-12	9.35	0.76

There mean age for no head lag was 3.46 months, for pull to sit 6.36 months and for pull to stand 9.35 months.

Correlation between HIE Stage and active tone

Table - 16

Stage	No head lag		Pull to sit		Pull to stand	
	Mean	SD	Mean	SD	Mean	SD
I	3.05	0.21	6.02	0.15	9.12	0.43
II	4.2	1.07	6.96	1.14	9.77	1.01
P	0.0001 Significant		0.0001 Significant		0.0001 Significant	

There was a significant correlation (p value 0.0001) established between HIE staging and active tone.

Table - 17

Correlation between mode of delivery and active tone

Mode of delivery	No head lag		Pull to sit		Pull to stand	
	Mean	SD	Mean	SD	Mean	SD
Labour naturale	3.5	0.93	6.39	0.84	9.42	0.87
Forceps	3.42	0.76	6.32	0.78	9.29	0.64
Vacuum	3.33	0.58	6.33	0.58	9.0	0.0
Breech	3.33	0.58	6.0	0.0	10.33	1.15
LSCS	3.43	0.88	6.34	0.82	9.2	0.53
P	0.9993 Not Significant		0.8315 Not Significant		0.1412 Not Significant	

There was no significant correlation established between mode of delivery and active tone.

Table - 18

Correlation between time of disappearance of primitive reflex and time of appearance of protective reflex with active tone

Correlation between	No head lag	Pull to sit	Pull to stand
Time of disappearance of primitive reflex	0.86 Correlated	0.75 Correlated	0.54 Correlated
Time of appearance of protective reflex	0.69 Correlated	0.57 Correlated	0.37 Not Correlated

The time of disappearance of primitive reflexes and appearance of primitive reflexes were correlated with the active tone.

DISCUSSION

Ideally all high risk babies need close surveillance throughout infancy. However in developing countries like ours with limited resources, poor transport system and rising cost of fuel this is not possible. It is essential to identify group of babies early on in infancy, who need close surveillance as far as neuro development is concerned.

Developmental tests in infancy are done basically for two reasons, firstly to diagnose deviant or delayed development so that early intervention can be started, and secondly for prediction of outcome.

Physicians who have put in a lot of effort to save a sick neonate would like to know as early as possible whether the infant is going to be neuro-developmentally normal.

The younger the child, the more limited is the range of abilities available for testing. Early developmental testing primarily measures biological functions and maturation of the neuro motor system. As the child enters the second year, development becomes increasingly influenced by a broader range of factors present in the surrounding environment. Hence, the pediatrician should have a clear understanding of what is being assessed, and what decisions he or she wants to take after knowing the results of this evaluation.

The other popularly used test in infancy is the one that originated in

France. This has been put in a structured, tabulated form my Amiel-Tison. Any study in neuro development has to be longitudinal in nature and assessment of tone is an Integral part of neuromotor examination.

Most conventional neurological assessment tests do not emphasize this aspect. Evaluation of tone must not only be quick and easy, but also standardized. Passive tone is assessed more accurately than active tone because it is independent of strength. It is determined by resting posture, angle of flexion, resistance to extension and passive recoil.

It also includes neurosensory evaluation, head growth and neurobehavior. However, this is a pure neuromotor test and does not take into consideration the mental development of the child at all. So if you need to assess the mental development, you have to take the help of some other method and this is the major limitation of this method.

Various tests have been used at different ages in infancy to predict outcome. However, clinical predictions are inherently probabilistic and can never be certain. No predictions regarding the diagnosis of cerebral palsy should be made on the basis of a single examination in view of the transience of tone abnormalities. Cautious optimism may be exercised in predicting outcome in high risk infants. Although structural recovery never occurs, functional recovery can occur due to early intervention.

We have shown that a normal neurological examination by Amiel Tison method at 3 months is an excellent predictor of normal outcome at twelve months.

Despite all the limitations and controversies surrounding present infant assessment tests, they do form an effective means of identifying infants with delayed development, so that early intervention can be started.

In this study two hundred and twenty seven full term infants were prospectively followed up for a period of one year. There were one hundred and thirty three infants in HIE stage I and seventy four cases in stage II. Twenty cases lost follow up. So only two hundred and seven infants came for all the four assessments.

Incidence of neurologic sequelae:

In this study severe neurological sequelae were noted in three out of seventy four babies i.e. 4% in this study. In a similar study conducted by Pelman JM et al[13] showed that perinatal hypoxic-ischemic cerebral injury, secondary to interruption of placental blood flow that results in cerebral palsy (CP), is a rare event. In Nelson et al[11] it was showed that fewer than 10% of children with CP had evidence of birth asphyxia.

Gozalez de Dios J,et al[14] in his study showed that neurologic manifestations was present in 25.6% of 156 term infants with perinatal asphyxia: 40 cases of hypoxic-ischemic encephalopathy (mild in 30,moderate in 5 and severe in 5). The incidence of hypoxic-ischemic encephalopathy was 1.19 cases per 100 full-term infants. The asphyxiated newborns were regularly assessed. Ten infants was lost to follow-up. The incidence of neurologic sequelae, in 115 asphyxiated full-term infants follow-up at least 12-24 months, was 16.5%. 4 cases of severe sequelae, 4 moderate and 11 mild. The overall asphyxia-related infant mortality rate was 0.87/1000 live births. The main sequelae detected at follow-up was motor disability, and other disabilities like mental retardation, epilepsy, sensorial defects, were in frequents. The incidence of cerebral palsy was 0.87/1000 live births, and 2.6% asphyxiated term neonates.

The lower incidence of CP noted in the present study may be due to exclusion of cases with severe encephalopathy or HIE stage III.

Robertson C, et al[9] in his study showed that a total of 167 term neonates with a diagnosis of hypoxic-ischemic encephalopathy (HIE) had detailed neurodevelopmental follow-up at 3.5 years of age. All 66 children with mild HIE were free from handicap; all seven with severe HIE were severely handicapped; and of the 94 with moderate HIE at birth, 21.3 per cent were handicapped.

Severity:

In this study no babies with HIE stage I had gross developmental delay few babies show mild delay. The incidence of delay in HIE stage II was found to be

15- 20 %. In that three babies show features of quadriplegic CP i.e. 4 %

Low birth weight and long treatment on a ventilator had a negative influence on the neuromotor behavior at 36 weeks' gestation
and white matter disturbances strongly affected the neuromotor parameters at 40 weeks(Katz Salamon,et al [15]).

Thornberg E, et al[16] in his study showed that all infants with severe HIE died or developed neurological damage. Half of the infants with moderate, and all of the infants with mild, HIE were reported to be normal at 18 months of age. A total of 0.3 per 1,000 live born infants died and 0.2 per 1,000 developed a neurological disability related to birth asphyxia.

Types of delay:

In this study delay in milestones occurred in 15- 20% of the children with HIE stage I and II. Among that only 3.4 % showed gross delay, the risk was highest in HIE stage II cases.

Bohr L[17], et al in his study showed the outcome for 1042 term infants born alive after likely intrapartum hypoxia-ischemia. Fifty-two percent had no sequelae, 8% had developmental delay without associated handicaps, 4% had a single handicap, 11% were multihandicapped and 14% were dead as a consequence of the intrapartum hypoxia-ischemia.

Visual disabilities:

In this study visual fixation and pursuit was present in 167 infants by three months, 38 infants by four to five months and in two infants not appeared till one year. Strabismus was noted in three babies in HIE stage II. It is concomitant type.

Sandfeld Nielson L, et al[18] in his study showed ophthalmic disorders in developmentally delayed children is increased if the child has CP, epilepsy, verified cerebral abnormalities or a genetic syndrome.

Hearing abnormalities:

No hearing abnormalities were noted in this study. In the majority of studies, birth asphyxia is not correlated with hearing loss in babies with complicated deliveries, prolonged artificial ventilation, the presence of severe hypoxic ischemic encephalopathy or persistent pulmonary hypertension are important factors. Perinatal hypoxia is more likely to cause a temporary hearing loss than a permanent one. Preterm babies are more vulnerable than term babies.(Borg L,et al[19])

Seizure disorder:

In this study recurrent convulsion in infancy was noted in three children, i.e.4%. All were from HIE stage II babies. Nunes ML, et al[20] in his study showed that neonatal seizures predominated in term newborns with perinatal asphyxia and an elevated perinatal mortality and post neonatal morbidity was observed. The follow up showed an increased risk for developing postnatal epilepsy and developmental delay.

Microcephaly:

In this study mean head circumference was comparatively lower in children with HIE stage II compared to stage I. Children with significantly less optimal head growth had developmental delay. Head circumference was normal in all children with normal neurological status.

Mercuri E et al[21] in his study showed at 12 months, microcephaly was present in 48% of the infants with HIE, compared with 3% of the controls. Suboptimal head growth was documented in 53% of the infants with HIE, compared with 3% of the controls. Suboptimal head growth predicted abnormal neurodevelopmental outcome with a sensitivity of 79% and a specificity of 78%, compared with the presence of microcephaly at 1 year of age, which had a sensitivity of only 65% and a specificity of 73%.

Risk factors:

The factors significantly associated with neuro developmental deficit were HIE stage II, early onset of convulsions, recurrent convulsions during infancy.

Sarnat and Sarnat staging:

In this study HIE stages showed marked correlations with neurodevelopmental deficit. None of the babies who had HIE stage I during neonatal period developed neurodevelopmental deficit. But HIE stage II were significantly associated with neurodevelopmental deficit.34 out of 74 babies (45.9%) with HIE II showed neurodevelopmental deficit by one year.

Disappearance of primitive reflexes:

In this study the mean age of disappearance of primitive reflexes were 3.78 months in the present study. In two cases of HIE stage II there were persistence of primitive reflexes. Those two cases had severe developmental delay and spasticity.

The combined examination of primitive reflexes and Postural reactions should be considered by the neurologist, as a simple but predictive screening test for the early identification of infants at risk for cerebral palsy. It is quick and easy to perform, both in nonhospital environments and in underdeveloped countries, where time and specific recourses are limited (Zafeiriou DI[22]).

Zafeiriou DI,et al[23] in his study concluded that from the results of one prospective and one retrospective study, it is clearly indicated that the absence of the plantar grasp reflex from 3 months of age and on correlates with the development of spastic cerebral palsy. The specific combination of presence or absence of specific primitive reflexes, postural reactions, or both may accurately predict a specific type of cerebral palsy or neuro developmental abnormality.

Appearance of protective reflexes:

In this study the mean age of appearance of protective reflexes were 9.76 months. In three cases protective reflexes were not appeared at the end of first year.

Tone assessment:

Passive tone is assessed more accurately than active tone because it is independent of strength. This study shows that the assessment of tone is a good predictor of outcome. The range of angle measurements were less in HIE stage II babies when compared to HIE stage I babies. This is one of the early predictor of neurological outcome. We used the goniometer to measure the angles in order to get a more objective evaluation, since visual measurements are more subjective.

Katz, et al[24] used the goniometer to measure the popliteal angle in CP children to evaluate hamstring muscle tightness.

Ten Berge, et al[25] in his study found that all interclass coefficients were lower in the CP group compared with healthy controls.

Thompson, et al[26] in his study showed that the popliteal angle is a widely used clinical means of assessing hamstring length in cerebral palsy patients.

Ma Fy, et al[27] used popliteal angle in his study on lengthening and transfer of hamstrings for a flexion deformity of the knee in children with bilateral cerebral palsy.

In a study by Mutlu, et al[28] it was concluded that the results from this study encourage the use of goniometric measurements in assessing children with spastic diplegic CP.

Kato, et al[29] in his study showed that evaluation of popliteal angle is useful for detection of infants with periventricular leukomalacia.

One should carefully interpret a tight Popiliteal angle in low birth weight infants during early infancy. Serial assessment of the Popiliteal angle is necessary before judging that a low birth weight infant has spastic cerebral palsy (Kato, et al[30]).

The babies with cerebral palsy had reduced adductor angles reflecting adductor spasm, and the baby with Werdnig Hoffman syndrome had an increased adductor angle reflecting increased adductor extensibility. The results provide a quantitative measurement of neurological maturation during infancy, and should complement the routine neurodevelopmental assessment (Quinn F,et al[31])

Waugh KG, et al[32] in his study used a goniometer to assess in 40 healthy, full-term newborns the following passive ranges of motion: hip extension, knee extension, ankle plantar flexion, ankle dorsiflexion, and the Popliteal angle. Every infant except one lacked full extension at both the hip and the knee. In active tone, the mean age of appearance of no head lag was 3.46 months, pull to sit 6.36 months and for pull to stand it was 9.35 months.

Samson JF,et al[33] in his study showed the best predictor of neuromotor behaviour at 7 years was the combination of outcome of
muscle power in shoulders and legs at 3 months and postural control at 12 months.

Gosselin J,et al[34] in his study concluded that the ATNAT which
takes 5 minutes to administer may be used in clinical setting as well as in research. Clustering of severe to mild neuro-cranial signs in the neonatal period permits identification of children who could benefit from early intervention.

Amiel-Tison’s Neurologic Evaluation of the Newborn and the Infant provides such a tool for use in the first year of life. This evaluation was developed to detect transient and permanent abnormalities in an infant's neuromotor development. Its main focus is to examine active and passive muscle tone. (McCarraher,et al[35])

LIMITATIONS

1. Hypoxic ischemic encephalopathy and hypoxic ischemic brain injury were not confirmed with biochemical analysis like scalp blood pH, umbilical cord blood pH or serum creatinine kinase brain bound.
2. Electro encephalogram and imaging studies were not taken into account.
3. Visual evoked potential, Otoacoustic emission and Brain Stem Response Audiometry was not done.
4. Regular follow up is needed for better results.
5. Further follow up is needed to detect minor neurological abnormalities.

CONCLUSION

1. In this study out of 207 cases followed no major evelopmental delay noted in HIE stage I cases and in HIE stage II delay in mile stones occurred in 15- 20% of the cases, among that only 3.4 % showed gross delay with features of spastic quadriplegic CP, the risk was highest in HIE stage II cases.
2. The risk factors for neuro developmental deficit observed in this study are sarnat and sarnat HIE stage II, presence of convulsions, delay in the disappearance of primitive reflexes and delay in appearance of protective reflexes.
3. It is inferred from this study that earliest age to detect neuro developmental abnormality is as early as three months.
4. The measurement of tone is an easy procedure done using goniometer. This study shows that the assessment of tone is a good predictor of outcome. The range of angle measurements were less in HIE stage II babies when compared to HIE stage I babies.
5. Early detection of neuro developmental abnormality is essential for early stimulation therapy.

RECOMMENDATIONS

1. By better obstetrical care birth asphyxia should be prevented.
2. Better neonatal care for asphyxiated babies to reduce the morbidity and mortality.
3. To keep a close watch on the neurodevelopment of the NICU graduates.
4. Amiel– Tison neurological assessment of tone using goniometer is easy to administer, may be used in clinical setting as well as in research.
5. Early detection and intervention is needed to reduce the severity of the disability.

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CASE SHEET PROFORMA

Name: Sex: In patient no:
Mother s name:
Father s name:
Address:

Date of birth: EDD:

Birth weight:
Length:
Head circumference:
Antenatal history:

Natal history:

Clinical examination:

1. Cry
2. Activity
3. Primitive reflexes:
    a) Moro
    b) Rooting & sucking
    c) Stepping and Placing

Sarnat staging: HIE I / HIE II

	1 month	3 months	6 months	9 months	12 months
HC					
Weight					
Length					

Vision:
Hearing:
Disappearance of primitive reflexes: months
Appearance of protective reflexes:

Milestones	Appearance	Delay	Not appeared
Social smile			
Head control			
Sitting			
Pincer grasp			
standing			

Evaluation of tone:

1. Passive tone

Angles	3months	6 months	9 months	12 months
1.Adductor				
2.Popileteal				
3.Dorsiflexion				
4.Scarf sign				

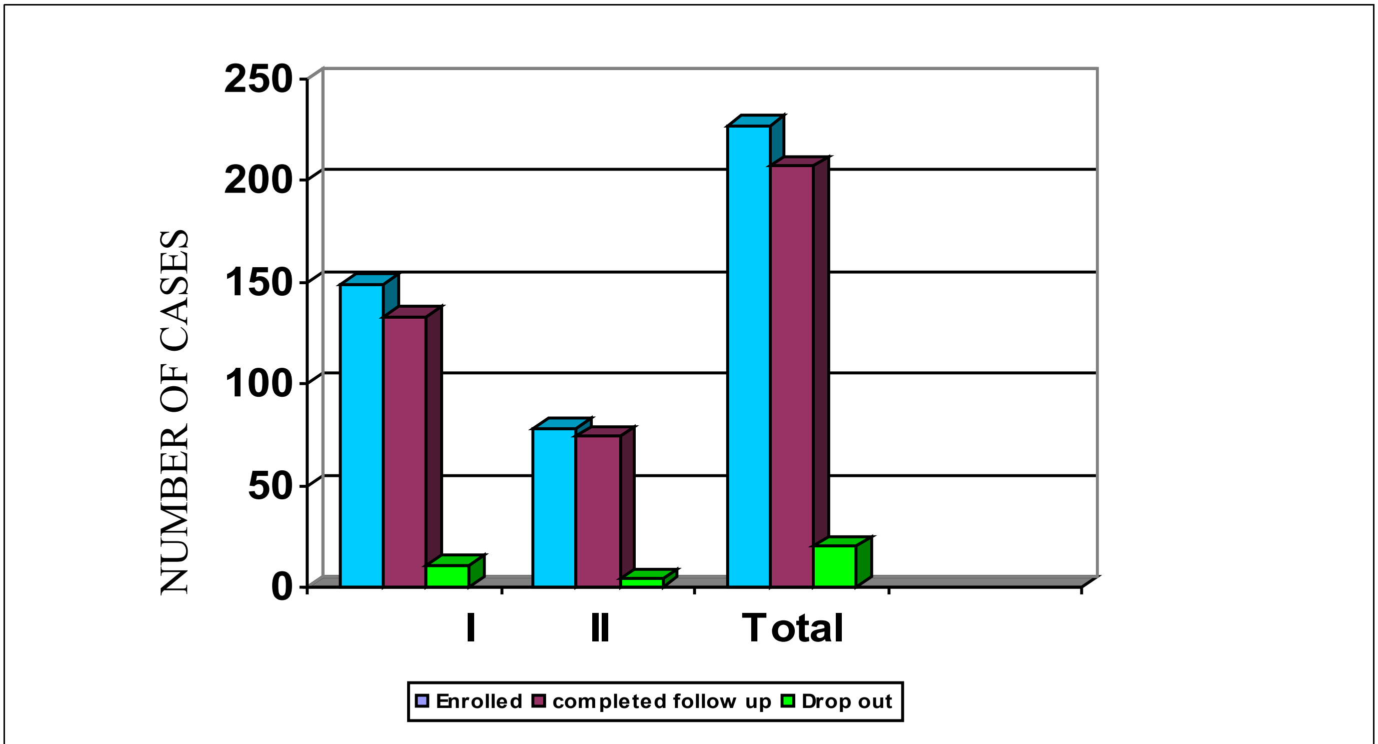
2.Active tone

	Months
No head lag	
Pull to sit	
Pull to stand	

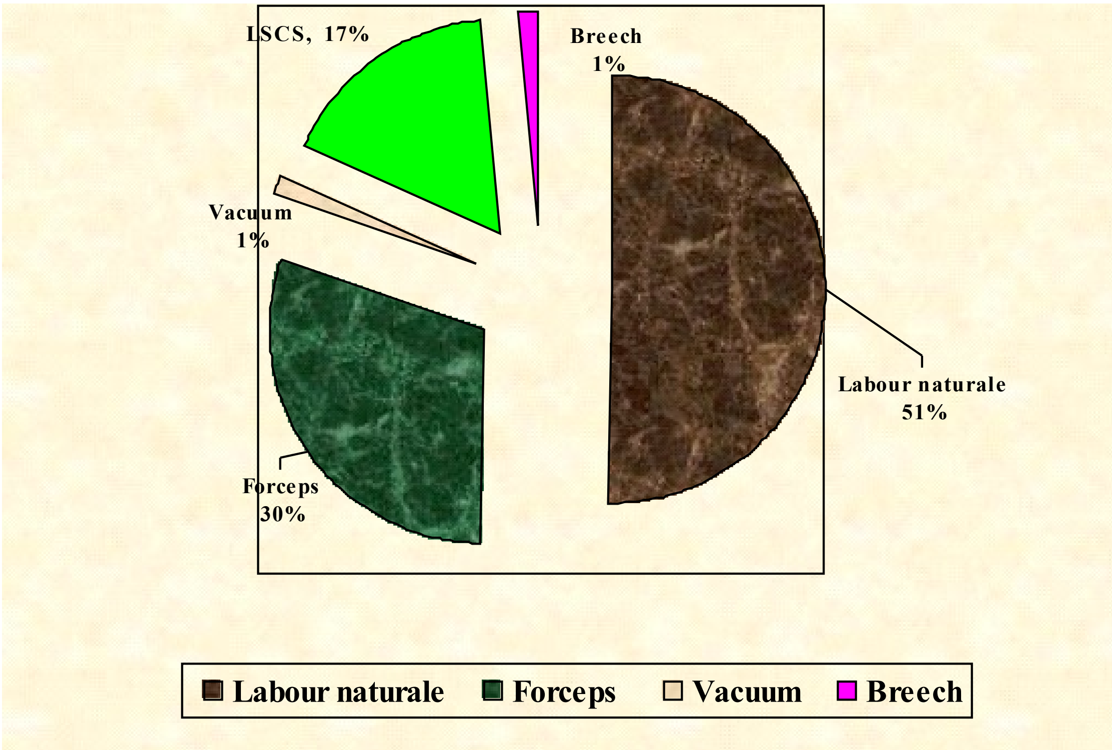
ABBREVATIONS

ATNAT	–	AMIEL TISON NEUROLOGICAL ASSESSMENT AT TERM
CO	–	CARDIAC OUTPUT
CFM	–	CEREBRAL FUNCTION MONITORING
CK-BB	–	CREATININE KINASE BRAIN FRACTION
CT	–	COMPUTED TOMOGRAPHY
EEG	–	ELECTRO ENCEPHALOGRAM
HIE	–	HYPOXIC ISCHEMIC ENCEPHALOPATHY
ICH &RC	–	INSTITUTE OF CHILD HEALTH AND RESEARCH CENTRE
LSCS	–	LOWER SEGMENT CESAREAN SECTION
MRI	–	MAGNETIC RESONANCE IMAGING
SD	–	STANDARD DEVIATION
AA	–	ADDUCTOR ANGLE
PA	–	POPLITEAL ANGLE
DF	–	DORSIFLEXION ANGLE
SS	–	SCARF SIGN
MM	–	MEDIAL TO MIDLINE
AT	–	AT MIDLINE
CM	–	CROSSES MIDLINE

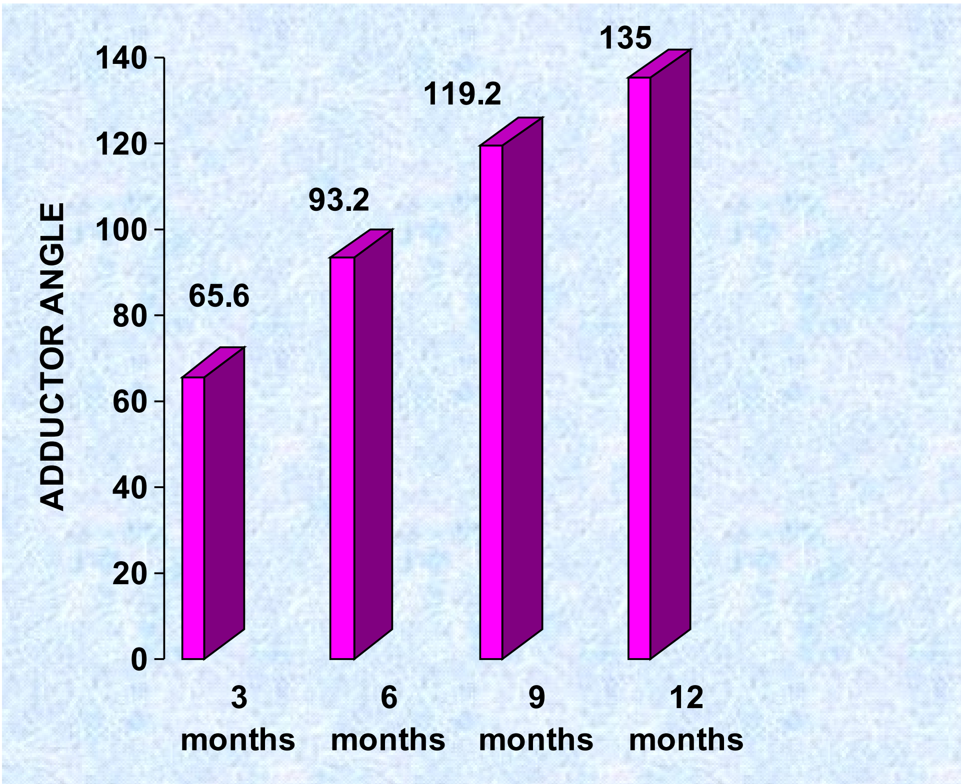
BASE LINE DATA



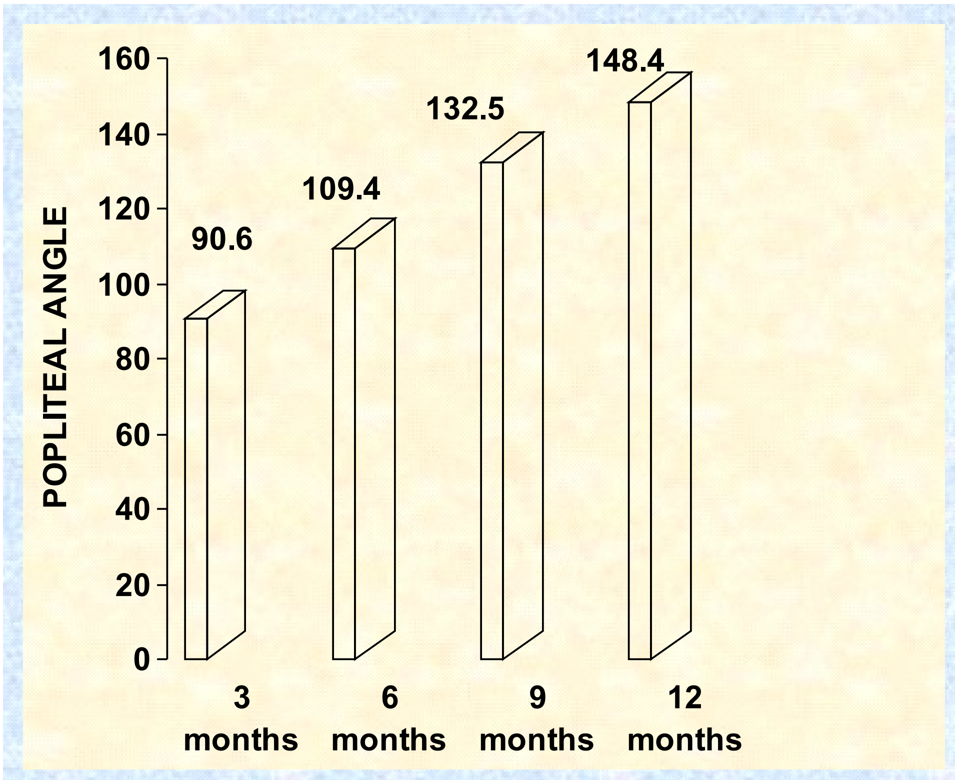
MODE OF DELIVERY



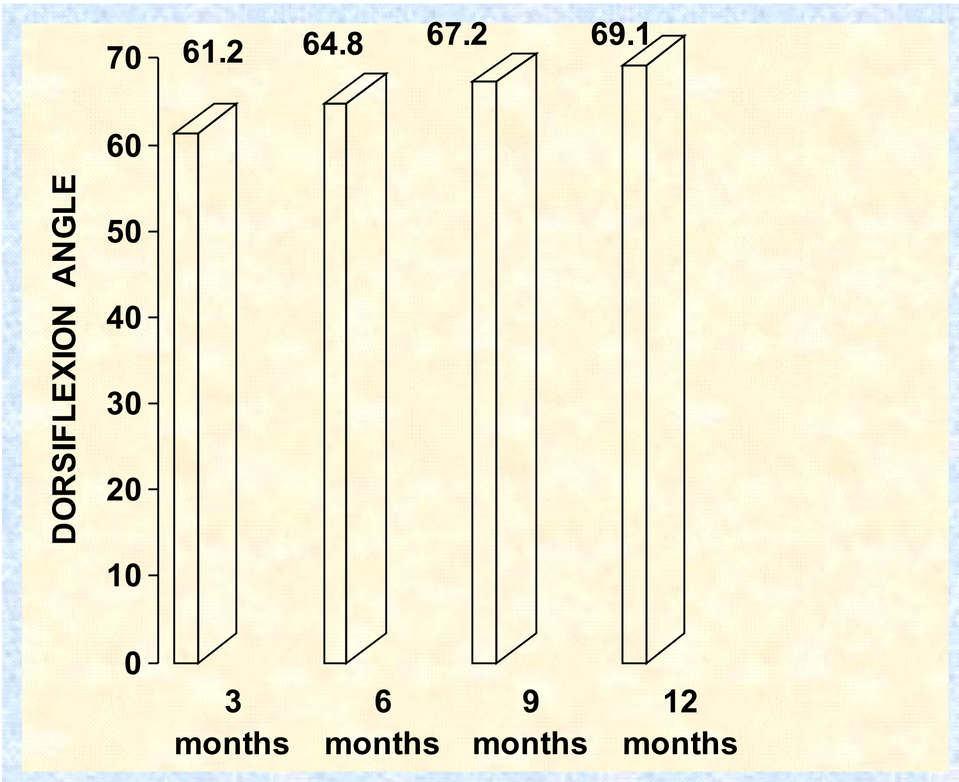
ADDUCTOR ANGLE



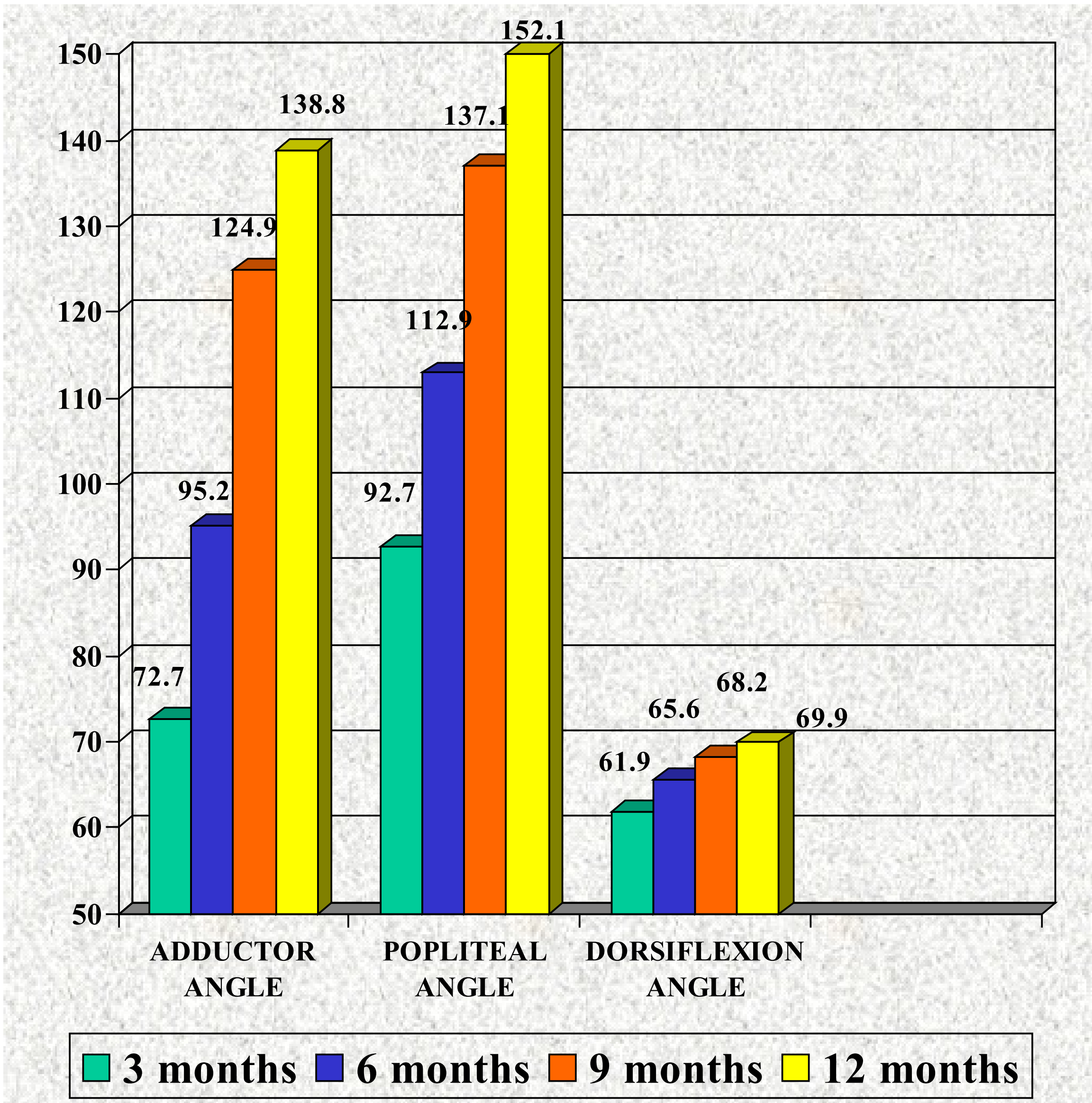
POPLITEAL ANGLE



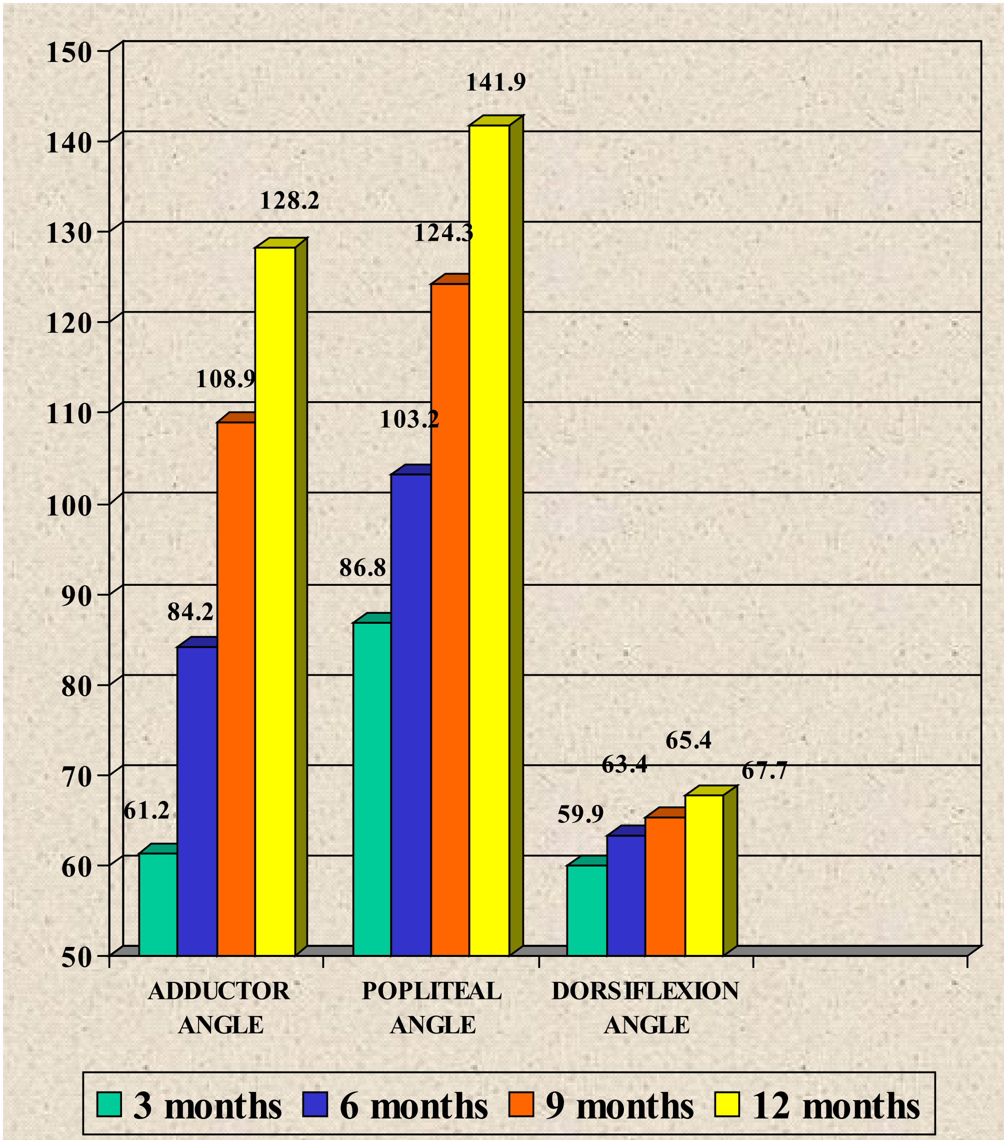
DORSIFLEXION ANGLE



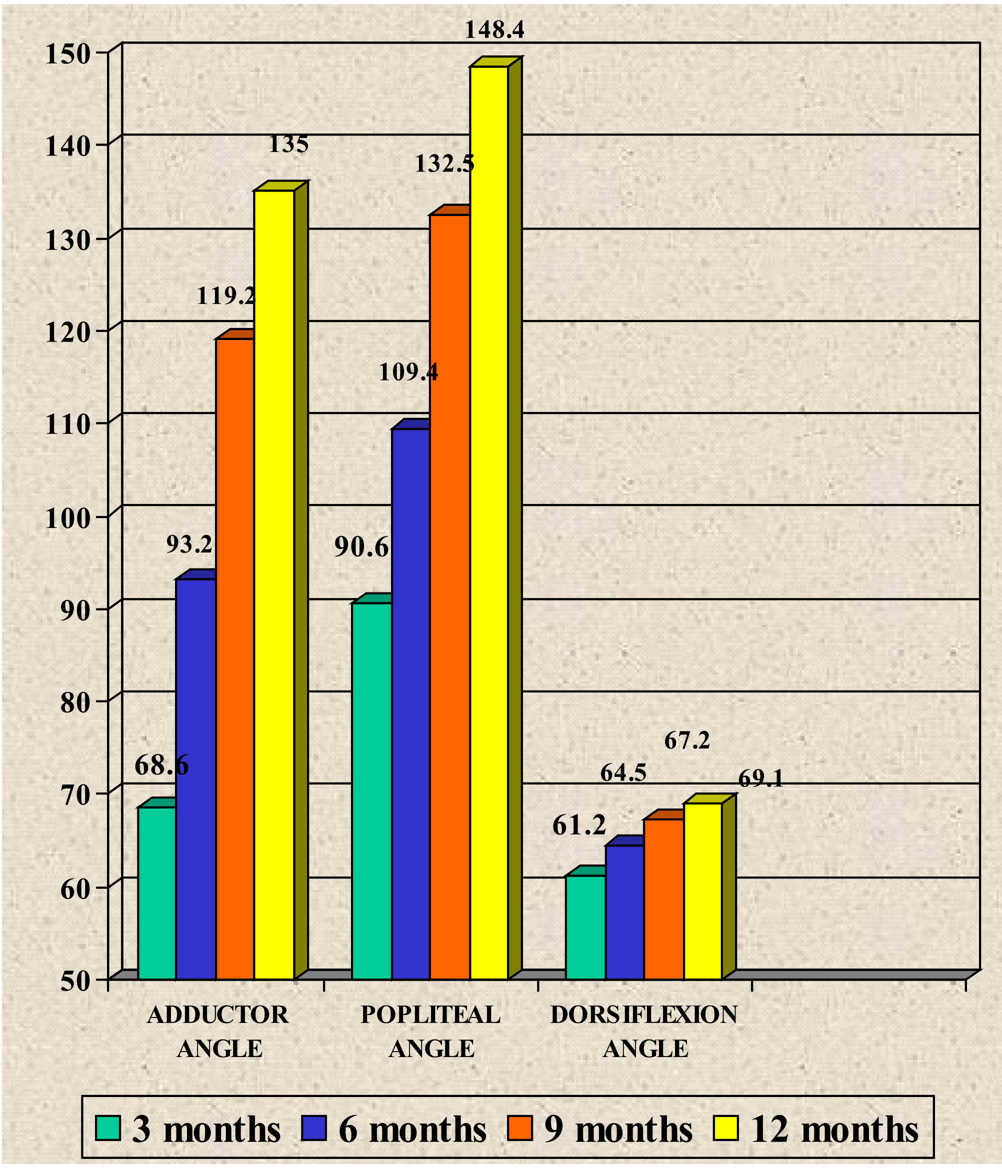
Measurement of angle at infancy (stage I cases no.133)



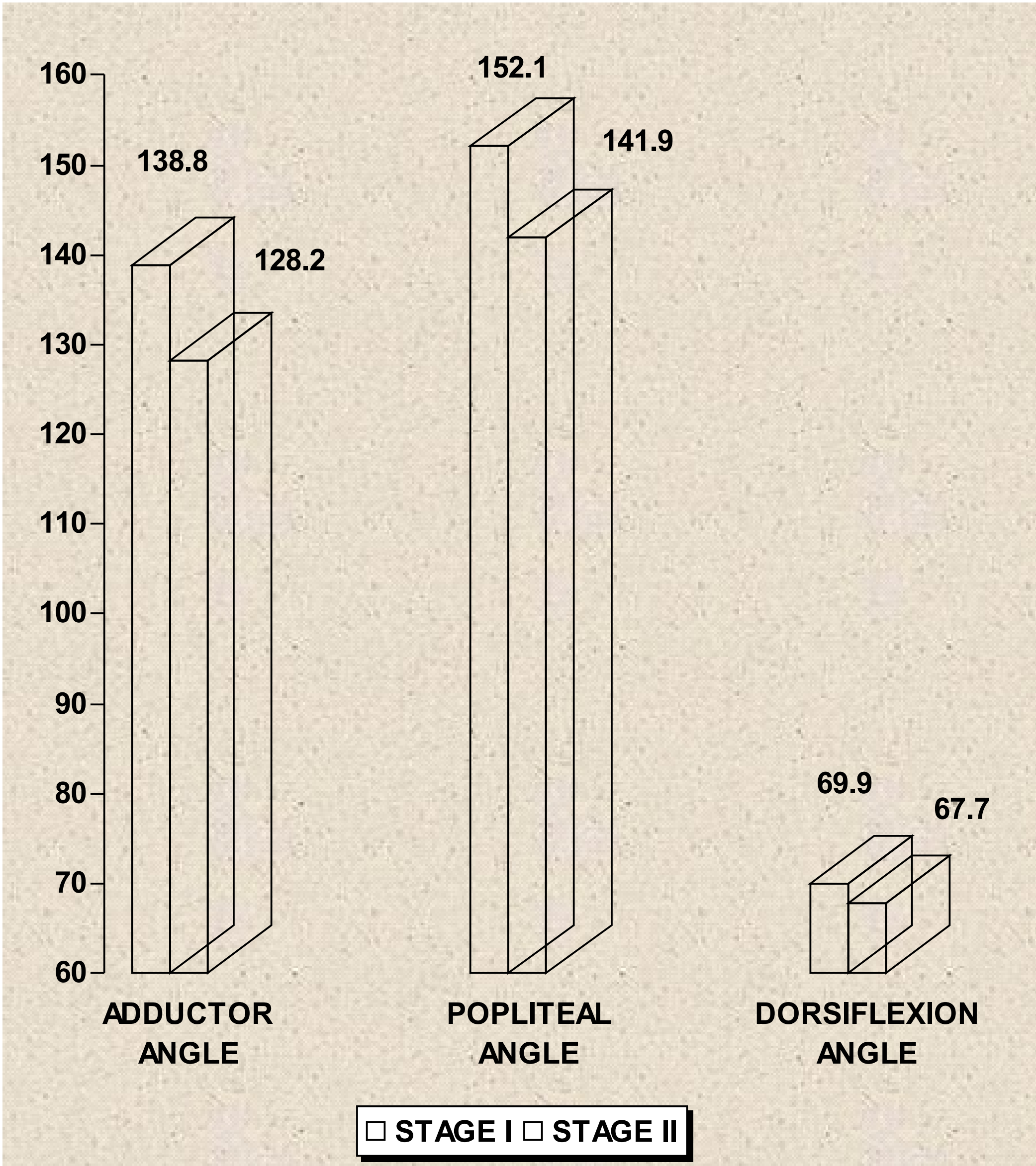
Measurement of angle at infancy (stage II cases no.74)



Measurement of angle at infancy (stage I & II cases no.207)



Correlation of HIE stage with passive tone



160
150
140
130
120
110
100
90
80
70
60
138.8
128.2
152.1
141.9
69.9
67.7
ADDUCTOR ANGLE
POPLITEAL ANGLE
DORSIFLEXION ANGLE
STAGE I
STAGE II

ADDUCTOR ANGLE

POPLITEAL ANGLE